can be classified as minor surgical procedures, little would be lost. Should some patients succumb to their disease, most of these would have lived their remaining days as "whole" women.

Reconstruction would be aided if the initial surgical operation were done through a transverse incision and if an attempt were made, were it appropriate, to save the nipple-areola complex by transplanting it as a graft to a site on the abdomen. Ths latter procedure should only be done after biopsy of the dermis under the nipple shows it to be tumor free. It could then be used in the reconstruction after the expanding procedures have been accomplished.[7]

Summary

Procedures are advocated for breast reconstruction in patients in whom radical and modified radical mastectomy has been done. This reconstruction includes the restoration of the nipple-areola complex as well as existing muscular defects. The approach has led to gratifying results in some patients for whom in the past reconstruction might have been deemed impossible with the more conventional techniques available.

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Angina Pectoris in Mitral Stenosis — Observations on a Mechanism

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A VARIETY of different chest pains, including angina pectoris, have been described in patients with mitral stenosis.[1,2] Several series have indicated a 5 to 15 percent incidence of typical effort angina pectoris in patients with mitral stenosis.[3-5] A wide range of explanations has been offered for this phenomenon and consensus does not exist; some consider the relationship only coincidental.[6] This report describes the case of a patient with severe mitral stenosis and pulmonary hypertension with effort angina. Findings on evaluation showed normal coronary arteries and pronounced hypotension with exercise. These data support the previously proposed mechanism of inadequate cardiac output with exercise resulting in inadequate coronary perfusion as a cause of angina pectoris in patients with mitral stenosis.

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Report of a Case

In a 43-year-old man there were murmurs consistent with valvular rheumatic heart disease known since 1960. The patient remained asymptomatic until July 1973 when atrial fibrillation and pulmonary edema developed. The rhythm reverted spontaneously to normal sinus rhythm, and the patient's clinical condition improved following diuresis. Subsequently, cardiac catheterization was carried out. The catheterization findings were consistent with calcific mitral stenosis with mild aortic insufficiency (see Table 1). Since only mild effort dyspnea was present at this point and commissurotomy was not felt to be feasible, it was elected to begin a regimen of digoxin and quinidine. In the ensuing months the effort dyspnea slowly increased. Also, the patient noted a substernal constriction or pressure which he clearly distinguished from dyspnea. This occurred in addition to dyspnea with more pronounced exertion and was relieved with five to ten minutes of rest.

In December 1974 the patient was admitted to hospital at Naval Regional Medical Center, Oakland, with subacute pulmonary edema. His condition improved notably with diuresis. Findings on physical examination after diuresis included blood pressure, 110/68 mm of mercury; pulse, 80 beats and regular; mean jugular venous pressure, 5 cm vertically above the sternal angle, carotid pulses normal, and chest clear to auscultation. On cardiac examination there were a right ventricular lift parasternally, increased first heart

TABLE 1.—*Cardiac Catheterization Data*

	Study of August 1973		*Study of January 1975 (before surgical operation)*	
Right atrial pressure	A wave (A) 16 mm Hg	mean 10	A 19 mm Hg	mean 12
	V wave (V) 12 mm Hg		V 12 mm Hg	
Pulmonary artery pressure	60/35 mm Hg	mean 45	110/33 mm Hg	mean 70
Pulmonary wedge pressure	A 28 mm Hg	mean 26	A 35 mm Hg	mean 32
	V 32 mm Hg		V 35 mm Hg	
Left ventricular pressure	100/10 mm Hg		118/6 mm Hg	
Aortic pressure	100/69 mm Hg		118/65 mm Hg	
Mean mitral gradient	16 mm Hg		25 mm Hg	
Cardiac index	1.84 L/min/m²		1.89 L/min/m²	
Mitral valve area	.8 cm²		.7 cm²	

Aortic insufficiency and mitral insufficiency were assessed as mild by angiography on both occasions.

sound (S_1), narrowly split second heart sound with pulmonic component increased, 2/6 apical diastolic rumble extending through to S_1 with presystolic accentuation. A 2/6 short decrescendo blowing diastolic murmur was present at the left sternal border and a 1/6 blowing holosystolic murmur at the apex. No hepatomegaly or edema was present.

Since the patient again felt relatively well and questioned the necessity of surgical operation, a treadmill test was carried out for functional evaluation. The patient completed three minutes of Stage I (1.7 mph, 10 percent grade) and 20 seconds of Stage II (2.5 mph, 12 percent grade) using the Bruce protocol.[7] He stopped because of dyspnea and substernal constriction. He noted no lightheadedness or alteration in consciousness and recovered to baseline within ten minutes. Blood pressure and heart rate are recorded in Table 2. Cardiac catheterization was repeated one week later (see Table 1). Findings on coronary angiography were completely normal.

Uncomplicated mitral valve replacement was carried out with a 29 Bjork Shiley prosthetic device one week after catheterization. Aortic insufficiency was assessed as mild at operation and the aortic valve left untouched. The postoperative course was uneventful, with pronounced improvement in effort tolerance and no further substernal pain with effort. Clinical evaluation three months postoperatively showed a normally functioning prosthetic valve with signs of reduced pulmonary hypertension. The aortic insufficiency murmur was barely audible. On repeat treadmill testing, the patient completed two and a half minutes of Stage III (3.4 mph, 14 percent grade) of the Bruce protocol before stopping because of leg fatigue primarily and also dyspnea. There was no angina, hypotension or ST-T abnormalities (see Table 2 and Figure 1).

TABLE 2.—*Treadmill Data*

	Blood Pressure in mm Hg	*Heart Rate Beats/Min.*
Before Surgical Operation		
Resting	120/80	73
Stage I, 2 min.	110/70	140
3 min.	50/35	142
After exercise, 1 min.	50/35	120
2 min.	100/60	112
After Surgical Operation		
Resting	120/80	69
Stage I, 3 min.	150/80	114
Stage II, 3 min.	160/80	129
Stage III, 2½ min.	180/85	156
After exercise, 1 min.	150/80	126

Discussion

The effort-related substernal constriction in this patient before surgical operation was typical of angina pectoris. Pronounced ST segment depression was recorded during the initial stress electrocardiogram but since he was receiving digoxin and quinidine, since the resting electrocardiogram was abnormal and since severe right ventricular hypertrophy was present, these repolarization changes are not necessarily reflective of myocardial ischemia. The lack of ST segment abnormalities on exercise testing postoperatively, despite continuation of digoxin and quinidine administration, does indicate an ischemic cause for the preoperative abnormalities. The clinical history, however, left no doubt that the patient was experiencing the symptom complex of angina pectoris.

Chest pain with mitral stenosis is relatively common and has been described in a number of reports since 1891.[8] Some of these descriptions have been of nonanginal pain, such as the hypercyanotic angina reported by Burgess and Ellis[1] and the prolonged chest pains described by Viar and Harrison.[2] Other reports have dealt with

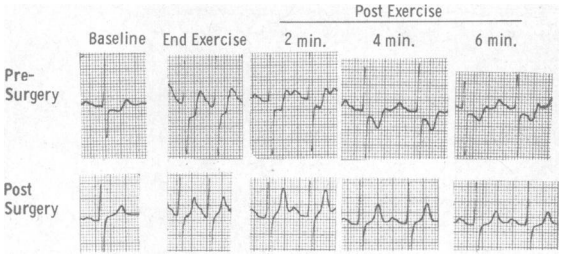

Figure 1.—Electrocardiographic complexes from Bruce Treadmill tests, V5. Patient was receiving digoxin and quinidine, in the same dose, during both exercise tests.

typical angina pectoris. Several studies in the British literature indicate a 5 to 15 percent incidence of typical angina pectoris in mitral stenosis.[3-5] These authors note the association of angina with severe mitral stenosis, severe pulmonary hypertension, or both. They attribute the angina to inadequate coronary blood flow due to inability to increase cardiac output with exercise. Stuckey further observed that a low fixed cardiac output with inadequate exercise increase is the common factor in other structural cardiac defects associated with angina, such as aortic stenosis, pulmonic stenosis or pulmonary hypertension. These data were collected before the general use of many current diagnostic techniques. Coronary atherosclerosis had to be excluded on presumptive clinical grounds (predominantly young female patients) or autopsy findings when available. Hemodynamic data with exercise were generally not available.

More recent studies have described sophisticated hemodynamic evaluation of exercise in mitral stenosis but do not include patients with angina. Bruce and co-workers reported hypotension with exercise in patients with mitral stenosis.[9] Frank and associates noted that coronary blood flow was dependent on central arterial pressure in these patients.[10] In those with mitral stenosis in whom hypotension developed with exercise there was a concomitant decrease in coronary blood flow. In cardiology texts there is no consensus on this question at present. Friedberg states "considering the frequent occurrence of mitral stenosis and angina pectoris as separate entities, the comparatively rare coexistence of the two conditions in the same patient must be explained as accidental, not causal."[6] Hurst concludes "the relevance of data supporting many of the conclusions seems to be open to question. In general, typical angina of effort in mitral stenosis should lead one to look for associated aortic valvular disease or coronary atherosclerosis."[11]

Other reports have suggested rheumatic coronary arteritis,[12] coronary artery emboli,[13] dilatation of the pulmonary artery[2] and compression of the left coronary artery between left atrium and pulmonary artery[14] as causes of angina in these patients. The data from this patient support the theory of Wood[5] and Stuckey.[3] With severe pulmonary hypertension and severe mitral stenosis, there were two obstructive lesions in series in the patient. The profound hypotension that occurred with exercise probably represents an extreme example of inability to maintain adequate cardiac output, commensurate with the increased demands of exercise. Coronary hypoperfusion almost certainly existed when angina developed since the arterial blood pressure was 50/35 mm of mercury. The data would suggest that the ischemic pain was generated in the severely hypertrophied right ventricle rather than the relatively normal and underfilled left ventricle. While the resting left ventricular pressure exceeded that of the right ventricle, this relationship was very likely reversed with exercise as systemic and left ventricular pressure fell. In any case the data indicate inadequate supply as a major abnormality in this patient's imbalanced myocardial oxygen supply and demand with exercise.

A single case will not finally resolve these questions. This case does support the most plausible explanation for this phenomenon. The currently available diagnostic techniques should allow definite resolution of this long standing controversy.

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